

Modern management of facial palsy:

a review of current literature

INTRODUCTION

Facial nerve dysfunction can severely affect a patient's quality of life. The human face is a focal point for communication and expression. The facial nerve carries motor, sensory, and parasympathetic fibres, so facial palsy results in both a functional and cosmetic impairment. Facial weakness secondary to upper motor neurone lesions will not be discussed in this article.

Facial nerve palsy is diagnosed upon clinical presentation with weakness of the facial muscles. There may be immobility of the brow, incomplete lid closure, drooping of the corner of the mouth, impaired closure of the lips, dry eye, hyperacusis, impaired taste, or pain around the ear.

There are many causes of unilateral facial palsy that should be considered, including idiopathic, traumatic, infective, neoplastic, congenital, and autoimmune (Box 1). Seventy per cent of facial nerve palsies are diagnosed as Bell's palsy¹ with 11–40 new cases per 100 000 each year.² Bell's palsy disproportionately attacks pregnant women and patients with respiratory tract illness. Bilateral facial palsy is far less common (2% of facial palsies) and typically represents a systemic disorder with multiple manifestations. Bell's palsy is also the most common diagnosis in childhood and accounts for 90% of facial paralysis. Up to 10% of patients with Bell's palsy will experience recurrence after a mean latency of 10 years.²

MANAGEMENT

The management of facial palsy is dictated by clinical assessment and working diagnosis. Facial motor neurone disturbance should only be considered as Bell's palsy after all other aetiologies have been excluded. Careful evaluation of the patient, particularly with respect to the history, otoscopy, and neurologic assessment, will differentiate a true Bell's palsy from other causes. A patient with Bell's palsy will typically present with acute onset, painless facial weakness (lower motor neurone distribution) with a normal

Box 1. Causes of facial nerve palsy

Trauma	<ul style="list-style-type: none">• Basal skull fractures• Birth trauma• Facial injuries• Otitic barotrauma
Neurologic	<ul style="list-style-type: none">• Brainstem infarction• Multiple sclerosis• Myasthenia gravis
Infection	<ul style="list-style-type: none">• Otitis media• Mastoiditis• Herpes zoster oticus• Mumps• Guillain-Barré syndrome• Syphilis• Leprosy• Lyme disease• Cat scratch
Metabolic	<ul style="list-style-type: none">• Diabetes mellitus• Pre-eclampsia• Acute porphyria
Neoplastic	<ul style="list-style-type: none">• Lymphoma• Tumours of the parotid gland• Tumours of the middle ear including cholesteatoma• Tumours of the petrosal bone• Tumours of the cerebellopontine angle including acoustic neuroma
Idiopathic	<ul style="list-style-type: none">• Bell's palsy• Melkersson-Rosenthal syndrome
Iatrogenic	<ul style="list-style-type: none">• Parotid surgery• Mastoid surgery
Autoimmune	<ul style="list-style-type: none">• Temporal arteritis• Systemic lupus erythematosus• Sarcoidosis• Thrombotic thrombocytopenic purpura

ear, nose, and throat (ENT) examination (Box 2). If the examiner is confident that there are no signs of an alternative diagnosis, then this patient may be managed in primary care.

Any patient whose presentation is atypical, be that an insidious and painful onset or clinical signs, such as otorrhoea and focal neurology, then a prompt and thorough work-

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Box 2. Bell's palsy

- Painless
- Acute
- Lower motor neurone (distribution of facial weakness)
- Systemically well
- Your examination is otherwise normal!

Box 3. House-Brackmann scale of facial nerve weakness

I	Normal
II	Mild weakness
III	Weak but eye closes
IV	Weak with incomplete eye closure
V	Flicker of movement
VI	No movement

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up should be carried out. In short, referral to ENT surgery is suggested in any patient whose presentation is atypical of Bell's palsy.

Grading the severity of weakness at presentation is recommended although it is of limited value. Grading is not prognostic and does not equate to stratified treatment protocol. The key observation is whether the weakness is complete or partial as this impacts on prognosis and the need for specialist referral. Many grading systems exist but the House-Brackmann scale (Box 2). Eyelid closure is an important observation as this carries the most significant functional consequence.

General

Eye care should be considered in every patient and is of paramount importance, as the eye is both unprotected and dry. A combination of artificial tears and ocular lubricant ointments will prevent drying and irritation. Tape across the eyelid will keep the eye closed at night. An ophthalmology opinion may be necessary in cases of long-term weakness or corneal damage.

Medical

Any reversible cause of facial palsy must be identified and treated immediately. If there is any suspicion as to the aetiology, then appropriate specialty referral should be made. Extensive clinical research on the treatment of Bell's palsy, including a number of recent, randomised controlled trials,^{3,4} has demonstrated that high dose oral steroids commenced within 72 hours of onset improves outcome. This is consistent with a 2010 Cochrane review.⁵ The authors recommend prednisolone 50 mg/day for 10 days. There is also good evidence that concomitant antivirals are of no benefit in the treatment of Bell's palsy. However, pain with vesicles in the ear canal or on the soft palate indicate zoster infection (Ramsey Hunt syndrome). This requires combined early treatment with steroids and antivirals because of potential disseminated viral disease and significantly worse prognosis for the facial palsy.

Surgical

Facial palsy secondary to an acute middle ear infection is likely to require grommet insertion with or without mastoidectomy. Surgical intervention may also be required if the nerve has been damaged by cholesteatoma or due to surgery or temporal bone fracture.

In summary, Bell's palsy does not routinely require referral to ENT surgery, particularly if the weakness is incomplete. Patients can be reassured but should be encouraged to seek follow-up if symptoms do not improve

over 2–3 weeks. In cases of complete or non-idiopathic facial palsy then referral to ENT is recommended. Bilateral facial weakness, recurrent facial weakness, or facial weakness that is not resolving after 3 months, are all indications for further investigation and should also be referred to ENT surgery. Referral to specialist centres (usually plastic surgery) for facial reanimation surgery may be appropriate in cases with long-standing residual weakness.

PROGNOSIS

The prognosis for recovery in Bell's palsy is influenced by a number of variables:

1. Rate of recovery: signs of improvement within 2 weeks favours a positive long-term outcome.⁶
2. Complete versus partial weakness: partial weakness favours complete resolution with full recovery in 94–99% of cases versus 75% with a complete paralysis.⁷
3. Age: younger patients tend to have a better prognosis.⁸
4. Steroids: commencement within 72 hours is proven to be beneficial.⁹

Key points include: uncharacteristic signs or symptoms should raise suspicion of an alternative diagnosis (for example Bell's palsy is painless whereas Ramsey Hunt syndrome is painful); eye protection is paramount if closure is impaired; and a partial weakness has a good prognosis and reassurance can be given.

CONCLUSION

Facial nerve palsy is commonly seen and requires prompt evaluation and diagnosis. The majority of cases can be managed with medical treatment alone but ENT or neurology referral should be considered in atypical cases. Despite most patients having a favourable outcome, there is a call for primary care physicians to appreciate the need for accurate diagnosis and the importance of early treatment, particularly if the diagnosis is anything other than Bell's palsy. Further research is required to identify those at risk of permanent paralysis and measures to prevent this.

Provenance

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